IN BRIEF

YUC and TAA1/TAR Proteins Function in the Same Pathway for Auxin Biosynthesis

Given the importance of auxin, it has been surprisingly difficult to elucidate its biosynthetic pathways in plants. Several Trp-dependent routes have been proposed, in which CYTOCHROME P450 79B2/B3 (CYP79B2/B3), YUCCA (YUC), and TRYPTOPHAN AMINOTRANSFERASE OF ARABIDOPSIS1/TRYPTOPHAN AMI-NOTRANSFERASE RELATED (TAA1/TAR) genes have been thought to act in separate pathways (reviewed in Zhao, 2010). Recent data have suggested that YUC and TAA1/ TAR genes may in fact be part of a single pathway, and new work from Stepanova et al. (pages 3961-3973) provides solid evidence for this using genetic, pharmacological, and biochemical approaches in Arabidopsis thaliana.

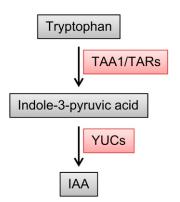
Stepanova et al. tested whether YUC1 functions in a pathway involving CYP79B2/ B3 or TAA1/TAR proteins. Overexpressing *YUC1* in a *cyp79b2/b3* mutant led to highauxin phenotypes, demonstrating that CYP79B2/B3 function is not required for YUC1-mediated auxin biosynthesis. Furthermore, *YUC1* overexpression led to additive phenotypes in the *superroot2* mutant, in which auxin biosynthesis via the CYP79B2/ B3 pathway is increased. Thus, YUC1 and CYP79B2/B3 appear not to operate in the same auxin biosynthetic pathway.

Arabidopsis encodes 11 YUC and three TAA1/TAR genes, and Stepanova et al. generated higher-order mutants, finding that reduced YUC function in backgrounds with already reduced TAA1/TAR function did not lead to additive phenotypes. Additionally, treatment with a newly described inhibitor of TAA1/TAR activity (He et al., pages 3944–3960) reversed the effects of YUC1 overexpression, supporting the idea that YUCs and TAA1/TARs function in the

same pathway. The authors also found that cells in which *TAA1* is normally expressed contain all of the necessary machinery for YUC1-associated auxin biosynthesis and that *YUC1* overexpression induced high-auxin phenotypes in a TAA1/TAR-dependent manner, adding further credence to the single YUC-TAA1/TAR pathway hypothesis.

Direct quantification of IAA and IAA metabolites gave evidence that overexpression of *YUC1* led to increased auxin biosynthetic activity in the wild-type background. This effect was absent in a TAA1/ TAR-impaired background, again consistent with YUCs functioning in the TAA1/ TAR pathway. The authors went on to show that pyruvate decarboxylases, which are key IAA biosynthetic enzymes in auxinproducing bacteria, are unlikely to play a role in auxin biosynthesis in *Arabidopsis*.

This work from Stepanova et al. nicely complements two newly published reports that use different mutant combinations and



A single pathway model for auxin (indole-3-acetic acid) biosynthesis via YUC and TAA1/TAR activities. (Adapted from Stepanova et al. [2011] and Won et al. [2011].)

different approaches to similarly demonstrate that YUCs function in the same pathway as TAA1/TARs, likely by converting indole-3pyruvic acid produced by TAA1/TARs into IAA (see figure; Mashiguchi et al., 2011; Won et al., 2011). Together, these studies represent an important step forward in understanding Trp-dependent auxin biosynthesis in plants.

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